EDITORIAL

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Delayed Gastric Emptying in Patients with Abnormal Gastroesophageal Reflux

It is now established that some patients with gastroesophageal reflux disease have delayed gastric emptying. It is believed that in this group of patients delayed gastric emptying is associated with a progressive dilatation of the proximal stomach which, in turn, shortens the length of the lower esophageal sphincter until it becomes incompetent – similar to the way distention of a balloon shortens its neck. With a shortened sphincter, and with greater amounts of solid and liquid materials in the stomach after meals because of its defective emptying, reflux occurs. Not surprisingly, these patients complain more often than those with normal gastric emptying of dyspepsia, postprandial distention, generalized bloating and abdominal pain, in addition to the usual symptoms of gastroesophageal reflux.[1]

However, because competency of the cardia is determined by many factors and because many patients with gastroesophageal reflux disease have normal gastric emptying, the importance of delayed gastric emptying in the pathogenesis of reflux (even in the generation of symptoms such as dyspepsia) has been questioned. Systematic measurement of gastric emptying in populations with abnormal reflux has shown that the rate of emptying does not necessarily correlate with the symptoms thought to be caused by delayed gastric emptying. Moreover, while most patients with abnormal reflux (with and without delayed gastric emptying) respond favorably to the use of proton pump inhibitors, very few respond solely to agents that promote gastric emptying. In fact, when combined with proton pump inhibitors, the additional benefit of prokinetic agents in the treatment of gastroesophageal reflux disease has been shown to be only marginal.[1] Even when the observations are restricted to patients with delayed gastric emptying and abnormal reflux, prokinetic agents do not ameliorate symptoms of reflux. For example, while cisapride normalized the rate of gastric emptying in 34 patients with abnormal reflux and delayed emptying of the stomach, heartburn and regurgitation remained unchanged.[2] Thus, while it makes sense to believe that delayed gastric emptying promotes gastroesophageal reflux, it is clear that enhancing gastric emptying in patients in whom it is defective has little impact on reflux symptoms.

The fact that gastroesophageal reflux disease does not improve with prokinetic agents, even when gastric emptying is normalized, brings into question the pathogenic role of defective emptying in the generation of reflux itself and of postprandial symptoms. For the surgeon, the problem is exacerbated by the following facts. First, it is known that after antireflux procedures, bloating and postprandial discomfort are seen in 20 to 40% of patients. While this problem is usually short-lived, a few patients continue to complain about it for many months and even years. Second, delayed gastric emptying postoperatively has been associated with postprandial distention, early satiety, dysphagia, and gas bloat in one study[3] and to the satisfactory outcome of a Nissen fundoplication in another.[4] Thus, the surgeon is concerned when considering antireflux surgery in patients with delayed gastric emptying and may be tempted to add a pyloroplasty or some other gastric drainage procedure with the hope of preventing delayed gastric emptying from worsening the side-effects of the antireflux procedure.

In this issue of the *Journal*, Bais et al.[5] studied 36 patients (26 with normal and 10 with delayed gastric emptying) before and after a Nissen fundoplication. The authors intended to determine both the effects of the operation on the rate of emptying and the impact that preexisting delayed gastric emptying might have on the outcome of the operation itself. The authors demonstrated that the Nissen fundoplication decreases the lag time between ingestion of food and the initiation of emptying and increases the rate of gastric emptying in all patients. Indeed, patients who had delayed emptying before the operation had normal values after it. More importantly, they found that the outcome of the operation, from a practical point of view, was the same whether the patient had delayed or normal gastric emptying preoperatively. Indeed, heartburn and regurgitation were controlled in both groups of patients and the side effects were quite similar and tended to subside within 12 months.

The fact that Nissen fundoplication increases the rate of gastric emptying has been previously reported.[6] This study, however, sheds some light on the mechanism. First, patients with defective emptying accumulated a greater proportion of the food in the fundus of the stomach for a longer time than did patients with normal emptying. This suggests that a defective function of the fundus (abnormal tone or too

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much receptive relaxation) may be responsible for delayed emptying by keeping the food away from the gastric pump (primarily the antrum). Second, they showed that after a Nissen fundoplication the defective accumulation in the fundus goes away and that distribution of food is exactly the same in both groups. With this new redistribution, the lag time between ingestion and initiation of emptying is shortened, and the total rate of emptying per hour is the same for both groups. Thus, it is clear that the defective fundus – now used by the fundoplication – is the culprit. The loss of gastric reservoir, normally a function of the fundus, explains the feeling of early satiety reported by most patients after this operation.

How can we apply this new knowledge, together with previous observations, showing that Nissen fundoplication increases the rate of gastric emptying? First, I agree with the author's main conclusion: the existence of delayed gastric emptying should not be a contraindication for a Nissen fundoplication. Second, those who do operations other than a Nissen fundoplication for the treatment of reflux may wish to consider a fundoplication in the group of patients found to have abnormal emptying, as this operation is the only one that clearly improves gastric emptying. Third, in patients with delayed emptying the surgeon should not advocate a pyloroplasty, but simply a fundoplication. This recommendation, while indirectly supported by the results of this study, stems from a number of other observations. First, pyloroplasty increases duodenogastric reflux, which may damage the gastric mucosa or worsen esophagitis if the cardia remains incompetent. Second, pyloroplasty without vagotomy is ineffective in the treatment of idiopathic or diabetic gastroparesis regardless of its effects on gastric emptying. Lastly, this study and the preponderance of evidence from other studies suggest that there is a good chance that gastric emptying will be normalized after a Nissen fundoplication. If that does not occur, there is always time to reassess the situation and devise a new strategy to deal with the problem.

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